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REVIEW

Primary megaureter: current trends in diagnosis and treatment

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Introduction

Reports of the spontaneous resolution of many cases of urinary tract dilatation detected before and after birth, even of severe degree, are increasing. Furthermore, differentiating urinary dilatation that is obstructive and requires surgery from that representing mere anatomical variation with no implication for renal function is a controversial issue in paediatric urology. The spontaneous resolution of hydroureteronephrosis and the controversy in identifying true obstruction caused urologists to re-evaluate their previous experience in the diagnosis and management of the problem of megaureter. In this article we try to resolve some of the disagreements about the diagnosis and management of this clinically important entity, by reviewing the most recent advances in this field.

Definition and classification

Much of the debate surrounding the diagnosis and treatment of 'megaureter' stems from the confusion in the nomenclature, as the term could be applied to any dilated or big (mega) ureter. Although the term megaureter means a single specific disease to some, it carries a mere connotation to others. Many classifications were proposed for megaureter, but clinicians should use the system that seems most simple and useful. The international classification of Smith *et al.* [1] is the most comprehensive (Table 1). In this scheme, megaureter may be obstructed, refluxing or unobstructed and not refluxing. Each of these major categories is subdivided into a primary and secondary group. In primary megaureter (PM) the cause is idiopathic, while secondary megaureter may be caused by urethral obstruction, BOO, neurogenic bladder, polyuria or infection. Although the classification is exhaustive, it is cumbersome. In general use, the term megaureter is not applied to a hydroureter caused by overt neuropathic bladder or typical BOO. Megaureter is usually reserved for conditions in which the bladder and bladder outlet are normal but the ureter is dilated to some extent. A more practical classification was given by King [2] as refluxing, obstructed, not refluxing, not obstructed, and both refluxing and obstructed. PM is an inherently compound term and usually includes obstructed PM and unobstructed PM. Secondary megaureter is caused by a variety of urological disorders, the diagnosis and management of which are beyond the scope of this review; the present focus is only on the subject of PM.

Demographics

PM is a common cause of obstructive uropathy among neonates and young children. In a series of 185 neonates with obstructive uropathy, vesico-ureteric junction (VUJ) obstruction was diagnosed in 44 (23%) [3]. PM is four times more common in boys than in girls and is bilateral in ≈ 25% of patients. Children presenting before 1 year old are more likely to have bilateral PM than are older patients [4]. The left ureter is involved 1.6–4.5 times more often than the right. In unilateral PM the contralateral kidney is either absent or dysplastic in 10–15% of patients. The condition is not known to be hereditary, but families with more than one member with PM have been described [2].

Histology

Several studies have examined the structure and ultrastructure of the normal and dilated ureter of childhood, using various techniques. Despite many differences, all of the reports agree that there are more connective tissue elements in megaureters, especially in

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Table 1 The international classification of megaureter

Class	*Primary*	*Secondary*
Obstructed	intrinsic ureteric obstruction	infravesical obstruction or extrinsic lesions
Refluxing	reflux is the only abnormality	associated with BOO or neurogenic bladder
Nonrefluxing/ unobstructed	idiopathic ureteric dilatation	polyuria (diabetes insipidus) or infection

the dilated portion [5–9]. In a recent study, Lee *et al.* [9] examined and compared the histology of ureteric smooth muscle and collagen in obstructive PM, refluxing megaureters and normal control ureters, using computer-assisted colour image analysis. The tissue matrix ratios (collagen : smooth muscle) were 0.52 in controls, 0.78 in obstructed PM and 1.99 in refluxing PM. At the ultrastructural level, Gosling and Dixon [7] found smooth muscle cells of the dilated ureter to contain fewer myofilaments and more organelles. They suggested that anomalous smooth muscle cells may be synthesizing collagen and that this could be the aetiology of the increased connective tissue.

Hofmann *et al.* [10] reported increased acetylcholinesterase activity in the juxtavesical segment of obstructed megaureters. Hertle and Nawrath [11] observed a tonic response to noradrenaline in isolated strips of obstructed megaureters. They ascribed this response to an increased fraction of intracellularly stored calcium in megaureters. Dixon *et al.* [12] showed dense nonadrenergic innervation in a smooth muscle collar surrounding the terminal ureter in cases of obstructed megaureter associated with ectopic ureteric insertion.

Aetiology and pathophysiology

Refluxing PM

Refluxing PM is caused by a short or absent intravesical ureter, congenital para-ureteric diverticulum or other derangement of the VUJ. Lee *et al.* [9] assumed that the marked increase in collagen and significant decrease in smooth muscle could be a major contributing factor in the pathogenesis of refluxing PM. They also expected that this group of patients would have a lower chance of surgical success even with a technically sound re-implantation.

There are a few patients with refluxing PM who have the megacystis-megaureter association, with the characteristics of bilateral hydroureteronephrosis, a large thin-walled bladder and bilateral high-grade VUR. The cause of the upper tract dilatation and the enlarged bladder is the constant recycling of massive amounts of refluxed urine [13]. Another special group of patients with dilated ureters are those with prune-belly syndrome, in which the ureterectasis may be caused by reflux or VUJ obstruction, or may be not refluxing and unobstructed, and categorized according to these characteristics.

In a few patients an element of obstruction may be combined with reflux. In a series of 400 refluxing renal units, Weiss and Lyton [14] found concomitant VUJ obstruction in nine (2%). A dysgenetic distal ureteric segment that not only fails to cope within the intramural tunnel but also has ineffective peristalsis is implicated. Identification is important because the management of obstruction often differs from that of reflux alone [15].

Nonrefluxing unobstructed PM

Most PM in neonates fall into this category [3]; there is neither reflux nor stenosis of the juxtavesical ureter, but the ureter is dilated beginning at a point just above the bladder. The cause of this phenomenon is unknown, but several theoretical explanations were given based on transitional (antenatal to postnatal) renal physiology and histo-anatomy of the developing ureter. Fetal urine production is 4–6 times greater before than after delivery, because of differences in renal vascular resistance, GFR and concentrating ability. This high outflow might contribute to ureteric dilatation in the absence of functionally significant obstruction, as is occasionally seen with diabetes insipidus [3]. Another contributing factor appears to be increased compliance of the fetal ureter because of differences in the deposition of type III collagen, elastin and other extracellular matrix proteins [16]. Finally, partial or transient anatomical or functional obstructions that spontaneously improve with postnatal development could contribute to proximal dilatation. Persistent fetal ureteric folds or delays in the development of normal peristalsis may represent such transient obstructions [3,17].

Obstructed PM

There is a general agreement that there is no true narrowing at the VUJ, but a functional obstruction arising from an aperistaltic juxtavesical segment 0.5–4 cm long that is unable to transport urine at acceptable rates. The pathogenesis of aperistalsis remains controversial and several explanations have been proposed. Excess collagen deposition has been a common finding [5–9]. In theory, increased matrix deposition alters cell-to-cell junctions and disrupts myoelectrical propagation and peristalsis [15]. Segmental changes of muscle cells were also highlighted in adynamic segments of obstructed PM, where there is atrophy of the inner longitudinal layer that conducts the peristaltic waves and hypertrophy of the outer circular layer that causes obstruction. Why the distal ureter is usually involved is explained by the embryological studies of Tanagho [18], who noted that the distal ureter is the last portion to develop its muscular coat and that early muscular differentiation is primarily of the circular muscles. The theory of segmental changes of muscle cells has been recently supported by Nicotina *et al.* [19], who detected TGF-β activity in the narrowed segments of PM but not in the dilated segments. TGF activity is not expected in the ureter postnatally, suggesting that there is some form of developmental arrest of the ureter at 20–28 weeks of gestation. This may explain the frequent spontaneous resolution of PM within the first 2 years of postnatal life, where the circular muscular pattern, which is typical of the fetal ureter, changes progressively into the double muscle layers of the full-term infant.

A band of circumferential tissue devoid of muscle in the most distal portion of the narrowed segment is also suggested as a cause of functional obstruction in some cases [20]. Tokunaka and Koyanagi [8] also suggested that a generously developed bulky peri-ureteric sheath appeared to contribute to obstruction in some cases of nonrefluxing megaureters. Dixon *et al.* [12] recently showed another cause of obstructive PM associated with ectopic ureteric insertion. There is a thick sleeve of muscle forming a continuous layer surrounding the muscle bundles of the terminal ureter. The muscle forming this outer layer is distinct in its histological appearance and arrangement from the muscle bundles which form the ureteric muscle of the VUJ. Furthermore, this muscle layer is very densely innervated by nonadrenergic (immunoreactive to dopamine beta-hydroxylase) nerves when compared with the smooth muscle coat of the ureter. The precise mechanism controlling this additional muscle layer remains unknown. This muscle collar has no continuity with either detrusor or ureteric muscle and thus myotonic influences such as those associated with ureteric peristalsis will not be transmitted to this region. Furthermore, the dense nonadrenergic innervation of this muscle collar might cause it to constrict inappropriately, impeding urinary flow, and ultimately lead to the development of megaureter.

Regardless of the cause, altered peristalsis prevents the free outflow of urine and functional obstruction results. Retrograde regurgitation occurs as successive boluses of urine are unable to traverse fully the aberrant distal segment. The degree of ureteric dilatation that results depends on the amount of urine forced to aggregate proximally because it is passed incompletely. This in turn is determined by the degree of distal obstruction and urinary output. This disruption in ureteric dynamics has obvious implications for the renal parenchyma if the collecting system is unable to ameliorate the proximal pressure that can develop [15].

Theoretically, it might be suspected that a compliant dilated ureter would provide a larger reservoir and hence a lower pressure buffering system than with dilatation caused by PUJ obstruction. Thus, it would be less likely for the dilatation caused by VUJ to transmit damaging pressure to the kidney. In contrast, a comparable PUJ obstruction would be likely have more of a direct damaging effect on the kidney with no more than the renal pelvis to diffuse the pressure.

Diagnosis

Megaureter is usually suspected through the clinical presentation and on grey-scale ultrasonography (US) before and after birth; the diagnosis is then confirmed by IVU and radioisotope renography. Reflux is usually diagnosed with no difficulty on voiding cysto-urethrography (VCUG). The primary nature of megaureter must be confirmed through the exclusion of secondary causes such as neurogenic and non-neurogenic voiding dysfunction, or infravesical obstruction caused by, e.g. PUV. The final and most difficult step in the diagnosis is to differentiate between an obstructed and unobstructed megaureter. Tests such as renal scintigraphy, Doppler ultrasonography (DUS) and pressure-flow studies (the Whitaker test) are useful for this and are summarized later.

Clinical presentation

The widespread use of obstetrical US with concomitant fetal screening have changed the age of presentation of congenital uropathies, including megaureter. Currently, about half of the cases are asymptomatic and discovered on prenatal US. Common symptoms are UTI, fever and abdominal pain. Microscopic haematuria is frequent and may occur in the absence of infection. This is presumably caused by the disruption of mucosal vessels of the ureter secondary to ureteric distension. Haematuria may also be a sign of calculus formation secondary to urinary stasis. It is not unusual for the dilated ureter to be noted at the time of abdominal surgery. Signs of renal failure are rare as presenting complaints.

Grey-scale US

Grey-scale US is the initial study obtained in any child in whom a urinary abnormality is suspected. It provides useful anatomical detail of the renal parenchyma, collecting system and bladder, and offers an invaluable baseline standard of the degree of hydroureteronephrosis that can be used in serial follow-up [15].

VCUG

Once ureteric dilatation is detected by US, VCUG is used to exclude reflux, and assess the quality of the bladder and urethra, in which neurogenic dysfunction, outlet obstruction or PUV are common causes of secondary megaureter. Reflux and obstruction may coexist in a small proportion of patients; in such instances, the reflux is often the most obvious finding and concomitant obstruction may not be considered unless the possibility of a dual lesion is considered. If a delayed film is obtained after the VCUG, poor drainage of one or both upper tracts may suggest associated VUJ obstruction. This can be confirmed or excluded by further evaluation.

IVU

IVU usually shows the pathognomonic configuration of a dilated distal ureteric spindle, a less dilated proximal ureter and a dilated or relatively normal-appearing renal pelvis. Caliectasis is either absent or mild. IVU is currently rarely used as an initial step in the diagnosis of PM because the functional data must be inferred. The substandard quality of this study also presents a problem with neonates because of renal immaturity and bowel gas. For these reasons, IVU should not be used before the age of 3–4 weeks and images taken 1 and 2 h after injection with contrast medium should be obtained. The anatomical detail provided by IVU is useful when the level of obstruction cannot be defined. A radioisotope renogram is preferred because it offers objective reproducible values for function and obstruction.

Methods for the differential diagnosis of obstructive vs unobstructive megaureter

The early diagnosis of congenital obstructive megaureter is an important opportunity to prevent renal damage from obstruction and infection, allow normal renal growth, and prevent symptoms caused by stasis and obstruction. During the past 20 years it has been recognized that urinary tract dilatation is not the same as urinary tract obstruction, which is true for cases involving the PUJ and the VUJ. Currently, there is no ‘gold standard’ for assessing renal obstruction with which to compare an individual case. The diagnosis in most cases is only possible by repeated investigations and comparing changes of the variables during a longer follow-up. Investigations including a renogram, DUS and the Whitaker test are helpful.

Renography

A renogram provides anatomical information together with objective reproducible variables of both function and obstruction. The most commonly used radionuclides are ^{99m}Tc-DTPA and -MAG3. For assessing obstruction, the renogram is used in several ways, the most common of which is the diuretic renogram (DR), detecting the pattern of the frusemide washout curve and the half-time drainage ($T_{1/2}$). However, several studies reported that many kidneys show stasis or a prolonged $T_{1/2}$, even though the follow-up showed adequate growth and function. This finding emphasizes that the diuretic curve and $T_{1/2}$ can be misleading, especially in the neonatal kidney. The extensive ureteric dilatation extending to the VUJ makes the timing of the administration of the diuretic and the determination of the area of interest largely empirical. Furthermore, the immature glomeruli of the new-born kidney are less sensitive to diuretics because the GFR is low. Therefore, whenever possible, it is preferable to defer the study for ≈ 3 months to allow glomerular maturation. In addition, false-positive results may be obtained if the child is dehydrated and if the single kidney GFR is < 15 mL/min.

In an attempt to circumvent the shortcomings of the diuretic curve and $T_{1/2}$, several modifications and techniques of renal scintigraphy were assessed, Deconvolution analysis of the renogram with the calculation of the parenchymal transit time was described by Whitfield *et al.* [21], and reported to be a more sensitive indicator of obstruction than the standard DR. However, other authors have shown little advantage over the standard DR and the complexity of the deconvolution analysis, particularly in children, has restricted its use.

English *et al.* [22] assessed a modified method of DR in which the diuretic was administered 15 min before the injection of radiopharmaceutical. This method is known as frusemide $-$ 15 (F-15), to distinguish it from the standard DR in which frusemide is given 20 min after the injection of the tracer and identified as $F + 20$. This modification is justified because the maximum effect of frusemide occurs 15–18 min after intravenous injection, and therefore results in maximum urinary flow rates at the start of the renogram. This helps to decrease the incidence of false-positive results in children with megaureter with grossly dilated or poorly functioning systems.

The extraction factor (EF) was described by Heyman and Duckett [23] as an estimate of single-kidney function in children during routine radionuclide renography with

^{99m}Tc-DTPA, and has been used by some investigators as a means of assessing clinically significant obstruction in children with megaureter [3,17]. The EF is defined as the percentage uptake of radionuclide by the kidneys 2–3 min after injection with isotope. The normal mean EF in the new-born is 1.5% by each kidney, with an increase to 2.5% occurring by the end of the first year of life. The correlation coefficient of the EF with GFR is 0.92. If renal function is normal by EF and/or equal to the contralateral undilated renal unit, the dilatation seen does not represent a functionally significant obstructive uropathy. Nevertheless, the EF has the disadvantage of a variability of $\approx 10\%$ from the GFR, and values for the percentage function in children aged 1–2 years have been defined incompletely.

Measuring individual renal function to detect a decrease in function has been used by some to show the presence of obstruction. The critical value of individual renal function below which obstruction is considered has varied among different studies. Values of $<30\%$ [24], $<35\%$ [25], or $<40\%$ [26] have been suggested (normal $50 \pm 5\%$). The failure of an expected improvement in renal function on further sequential radionuclide studies could be an indicator of the presence of obstruction.

Doppler ultrasonography

DUS with a measurement of renal resistive index (RI) was recently introduced as a noninvasive method of diagnosing obstructive uropathy [27]. Although an RI of 0.70 is accepted by most investigators as an upper level of normal in the adult population, more work is needed to establish an accepted standard of normal and abnormal for paediatric renal RI. It has been shown that the RI is age-dependent and is commonly >0.70 in healthy children in the first year of life [28]. A normal RI in this setting should be of value, arguing against obstruction in the presence of a dilated collecting system. An RI threshold of 0.70 gave a sensitivity of 82%, a specificity of 63% and an overall accuracy of 76% in children with obstructive uropathy [29].

Several methods have been used to increase the diagnostic accuracy of RI. The infusion of normal saline and administration of frusemide significantly decreased the RI of unobstructed kidneys and increased the RI of obstructed units [29]. This divergent response could help to identify obstruction. The calculation of the difference between the RI of obstructed and unobstructed kidneys (ΔRI) strengthens the diagnosis. Values of ΔRI of >0.06–0.10 are diagnostic of obstruction. However, comparison with the contralateral kidney is not applicable in patients with solitary kidneys or bilateral obstruction. Recent reports showed a good correlation between the RI and DR [30,31]. Therefore, the RI could be used for monitoring the dilated collecting systems under observation, obviating the need for frequent radioisotope scanning.

The Whitaker test

The Whitaker test is currently rarely used in the diagnosis of children with megaureter because it is invasive, does not measure function, submits the collecting system to unphysiological rates of flow and has a variable correlation with functional studies such as the DR. Moreover, the intrinsic urine output of the kidney contributes an unknown volume to the total amount of fluid being perfused, and the potential for false-positive results should be considered, particularly in neonates with a high urine output. In addition, when an obstruction is present in a hugely dilated system, the intrapelvic pressure will not increase until after the collecting system is filled completely. Thus, there is the potential for recording a falsely low pressure if there is a leakage of fluid outside the collecting system or if the study is terminated before the pelvicalyceal system is filled completely. These problems may be overcome by using fluoroscopy during the study.

Concomitant PUJ obstruction

Concomitant ipsilateral PUJ obstruction was seen in six of 47 (13%) infants with obstructive PM in one series [32]; McGarth *et al.* [33] reported a similar association in 14 children. The preoperative diagnosis of both pathological conditions may be difficult. When PUJ obstruction is present, the ipsilateral ureter is seldom seen on IVU. The diagnosis is not made until a retrograde ureterogram is taken when the child is under anaesthesia for a pyeloplasty or when the ureter is seen to be dilated at the time of the pyeloplasty. A preoperative renal scan is more helpful in this regard, as the obstructed ureter usually fills and is visualized as the obstructed pelvis drains [2].

Treatment

The treatment of refluxing PM and nonrefluxing unobstructed PM is fairly well established. Nevertheless, controversy remains about the management of asymptomatic patients with obstructed PM.

Refluxing PM

With the advent of antenatal US the management of refluxing PM has changed in the past few years. The previous recommendation for surgery in newborns and infants with grades 4 and 5 reflux is obsolete. Instead, medical management is appropriate during infancy and is continued if there is a trend to resolution. Otherwise, surgery remains the recommendation for persistent high-grade reflux in older children and adults [15].

Nonrefluxing unobstructed PM

Most patients who present with PM, particularly older children or adults, clearly have the unobstructed variety. Most of these patients remain asymptomatic without surgery, and expectant management results in an improvement in the degree of urinary tract dilatation and no deterioration of renal function.

Obstructed PM

There is a general agreement among urologists that when significant obstruction is present there is a need for early surgical correction to preserve renal function. However, presently there is no ideal method for assessing urinary tract obstruction, particularly in neonates and infants. Therefore, the therapeutic recommendations for neonates with obstructed PM remain controversial. Several published series supported early surgical repair [32,34,35]. The goals of early surgery are to minimize renal damage from obstruction, to maximize the growth potential of the affected kidney and to prevent problems from PM, especially infection and stone formation. Peters *et al.* [32] reported on 42 infants with obstructed PM operated upon before 8 months of age. The function of the obstructed kidney improved in all patients. Similarly, Mollard *et al.* [34] reported excellent results in 36 of 39 neonates with obstructive PM treated by early surgery. Nevertheless, in this approach the authors were criticised for having largely relied on drainage curves of the renal scan or on IVU to conclude that the systems were obstructed. Moreover, it was not known whether the

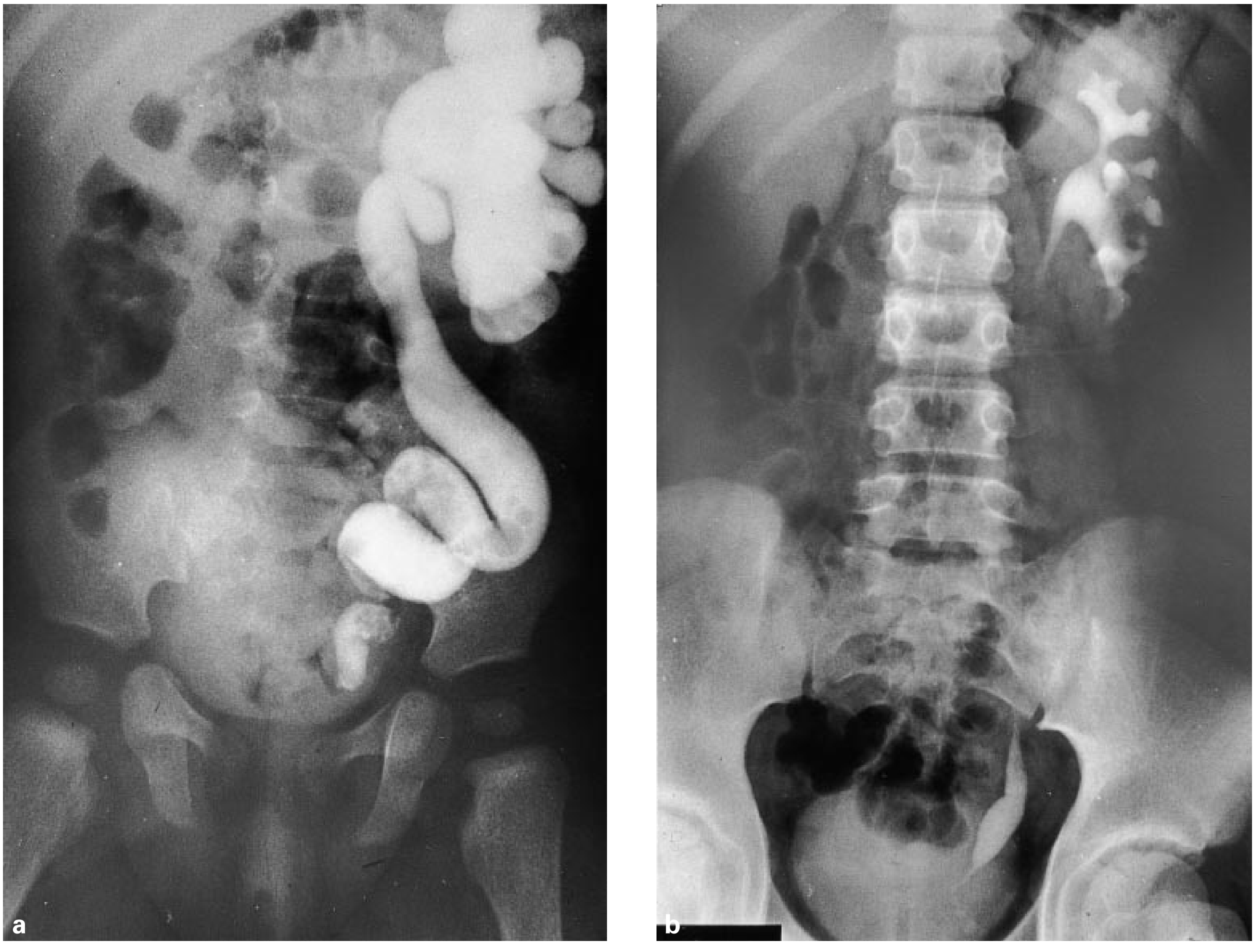

Fig. 1. a, IVU in a 3-month-old girl shows all the typical signs of a primary megaureter of the left solitary kidney. Renal function was normal, while the DR showed poor drainage ($T_{1/2} > 30$ min). She was treated conservatively with antibiotic prophylaxis (until the age of 5 years) and closely followed. **b,** IVU of the same girl at the age of 9 years, showing an almost normal renal configuration, although the distal ureter is still slightly dilated. This distal segment always is the last to become normal. Renal function remained normal, with no UTIs and the $T_{1/2}$ became normal.

improvement in renal function detected by follow-up renography was a result of early surgery or of the natural increase in renal function in early postnatal life. Furthermore, in the neonatal period the complication rate is higher for reimplantation of a dilated ureter, especially into a small bladder. In the series of Peters *et al.* [32], the rate of reoperation for reflux or obstruction was 12% (five of 42).

During the past 10 years there has been an increasing trend towards conservative management [3,17,36–38]. In this approach three main variables need to be monitored, i.e. symptoms, imaging and antibiotic prophylaxis. Symptoms are relatively straightforward to follow and surgery is indicated if there is persistent pain, breakthrough pyelonephritis or calculi. The frequency of imaging is greater at an earlier age, alternating between renal scintigraphy, RI and IVU. As the urinary tract dilatation stabilizes, the imaging interval increases. Renal function is best assessed by renal scintigraphy and surgery is indicated if there is a decrease or lack of the expected increase in the percentage of renal function of the developing neonatal kidney with time.

Antibiotic prophylaxis must be begun routinely soon after delivery in infants with prenatally detected hydroureteronephrosis. Prophylaxis is continued after confirming the diagnosis of an obstructed PM. When surgical repair is undertaken prophylaxis is continued until the obstruction is relieved and no reflux is detected. Oral penicillin-G (20 000 IU/kg), amoxicillin (15 mg/kg) or trimethoprim (2 mg/kg) once a day are commonly used.

Keating *et al.* [3] followed 44 hydronephrotic renal units in neonates who were diagnosed as having obstructed PM on the basis of renal scans alone soon after birth. None had deterioration of renal function over time. These authors concluded that obstructed PM diagnosed at birth because of antenatal hydronephrosis might be a distinct entity which carry little risk of subsequent deterioration in renal function. Baskin *et al.* [17] continued the initial study of Keating *et al.* [3] and reported on the long-term follow-up of the patients from the initial study who were managed with no surgery. There was no deterioration in renal function, or any episodes of urinary calculi or pyelonephritis during the follow-up, which extended beyond 12 years. Therefore, the authors proposed a more appropriate name for this entity, 'primary dilated megaureter', as obstruction implies the need for operative intervention. Figure 1 is an example of successful conservative treatment of a case of PM diagnosed as obstructed on the basis of DR.

Despite these gratifying results, some authors reported a failure rate for conservative treatment of 84% [35]. As there is no 'gold standard' for assessing renal obstruction to differentiate between obstructed and unobstructed PM, controversy over how best to manage asymptomatic patients with obstructed PM will continue. Nevertheless, the uncertainty in objective assessments of renal obstruction should not be a justification for inaction.

Principles of surgical treatment

Resection of the distal ureter only is rarely sufficient to permit reimplantation, and some means of reducing the calibre of the distal ureter is necessary. Two methods can be used to remodel megaureters. Formal ureteric excisional tapering is preferred for severely dilated ureters or those that are markedly thickened. Plication of the distal ureter is used when there is less dilatation, but may produce a bulky and stiff ureteric segment. Nonetheless, the frequency of postoperative reflux is no different between these methods [32]. Reduction of the calibre of the distal and mid ureter previously tended to be more radical; the recent trend has been to limit this reduction to that section of ureter necessary to achieve an adequate length of intravesical tunnel [32].

Remodelled megaureters have generally been reimplanted using a variety of well described surgical techniques, including the standard cross-trigonal, Leadbetter-type techniques or extravesical submucosal tunnel repairs. Success in reimplanting remodelled megaureters, regardless of technique, is not as high as with undilated ureters; the incidence of postoperative reflux is up to 10%. We have recently introduced a new principle for the extravesical reimplantation of dilated ureters (the extravesical seromuscular tunnel), in which the distal ureter is completely surrounded by detrusor muscle over its whole circumference [39]. We assume that this principle is more effective in preventing reflux than the standard submucosal tunnel techniques. Nevertheless, this assumption needs to be confirmed by other clinical studies.

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